POSTTRAUMATIC STRESS DISORDER: Etiology, Epidemiology, and Treatment Outcome

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■ **Abstract** Posttraumatic stress disorder (PTSD) results from exposure to a traumatic event that poses actual or threatened death or injury and produces intense fear, helplessness, or horror. U.S. population surveys reveal lifetime PTSD prevalence rates of 7% to 8%. Potential reasons for varying prevalence rates across gender, cultures, and samples exposed to different traumas are discussed. Drawing upon a conditioning model of PTSD, we review risk factors for PTSD, including pre-existing individual-based factors, features of the traumatic event, and posttrauma social support. Characteristics of the trauma, particularly peritraumatic response and related cognitions, and posttrauma social support appear to confer the greatest risk for PTSD. Further work is needed to disentangle the interrelationships among these factors and elucidate the underlying mechanisms. Based upon existing treatment outcome studies, we recommend use of exposure therapies and anxiety management training as first-line treatment for PTSD. Among psychopharmacological treatments, selective serotonin reuptake inhibitors evidence the strongest treatment effects, yet these effects are modest compared with psychological treatments.

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HISTORY AND BACKGROUND

The psychological consequences of trauma exposure are well recognized throughout history. Eloquent descriptions of trauma's sequelae can be found in references to the Civil War (e.g., Stephen Crane's description in *The Red Badge of Courage*), the 1666 Great Fire of London (Daly 1983), and even the Trojan War (e.g., the story of Ulysses in Homer's *Iliad*). Anxiety as a specific response to severe stressors has also long been observed as a separate clinical entity. Oppenheim (1892; cited by Kraepelin 1896) labeled this response "traumatic neurosis" and Kraepelin (1896) labeled it "Schreckneurose" (i.e., fright neurosis).

Despite these observations, the scientific study of traumatic responses is a recent enterprise. After World Wars I and II, studies of psychological distress among combatants and concentration camp survivors set the stage for the recognition of the long-term negative effects of trauma exposure. Terms such as "shell shock," "combat fatigue," and "war neurosis" all seemed to capture the essence of war-related posttraumatic stress disorder (PTSD), but the psychological impact of other traumatic life stressors received greater recognition in the research literature when Ann Burgess observed the impact of rape on women (Burgess & Holmstrom 1974). Once advocates for rape survivors and Vietnam veterans teamed with scholars who had studied and treated World War II veterans and concentration camp survivors, the diagnosis of PTSD emerged.

In view of the early recognition of traumatic neurosis and related emotional disorder, it is surprising that controversy regarding its validity emerged during the 1970s and 1980s (Figley 1978, Goodwin & Guze 1984). Although much of the initial controversy about the validity of PTSD has ceased, the nature of the disorder still instigates considerable debate in the field (see Brewin 2003). Conceptual issues have arisen regarding the definition and assessment of traumatic events, whether symptoms alone should define the disorder, and the concern that PTSD is a product of social construction (McNally 2004).

In its current conception, the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV; Am. Psychiatr. Assoc. 1994) defines PTSD as necessarily stemming from an event in which one is exposed to serious threat of injury or death, then experiences extreme fear, helplessness, or horror. Three symptom clusters define the disorder. In addition to recurrent and intrusive recollections and dreams of the event, the re-experiencing cluster includes distress and physiological reactivity upon exposure to trauma cues and flashback episodes wherein an individual

experiences a recurrence of at least a portion of the trauma. Hyperarousal symptoms are characterized by an enhanced startle reaction and difficulty sleeping, concentrating, and controlling anger, as well as hypervigilance for danger. Extreme distress and avoidance of cues or reminders of the trauma, as well as an inability to remember aspects of the event also can accompany this disorder. Additional symptoms falling within the avoidance cluster involve emotional numbing, including feelings of detachment from others; decreased interest in activities; an inability to feel any positive emotions such as love, contentment, satisfaction, or happiness; and a sense of a foreshortened future.

The interpersonal, psychosocial, physical health, and societal consequences of PTSD are considerable. Those with PTSD are more likely to divorce, report trouble raising their children, engage in intimate partner aggression, experience depression and other psychological problems, report poorer life satisfaction and physical health problems, become involved with the legal system, earn less, and change jobs frequently (Jordan et al. 1992, Koss et al 1991, Kulka et al. 1990, Schnurr & Green 2004, Walker et al. 2003). These findings suggest that PTSD constitutes a major problem for the public health of this nation and the world, and highlight the importance of prevention and intervention efforts. As with many conditions, we've accomplished more in terms of treatment than in prevention. This review outlines our achievements to date in the understanding of the characteristics of PTSD and its prevalence, course, and treatment. Further, we provide a heuristic model for understanding the development of PTSD while specifying future directions for scientific work.

PREVALENCE AND COURSE

When initially delineated in the DSM-III, PTSD was considered a relatively rare condition, and traumatic events were considered extreme life stressors that were outside the range of normal human experience (Am. Psychiatr. Assoc. 1980). Since the incorporation of PTSD into the diagnostic nomenclature, epidemiological studies have documented high prevalence rates of exposure to traumatic events in the general population and confirmed that PTSD occurs following a wide range of extreme life events. Most important, though, are the consistent findings indicating that although exposure to potentially traumatic events is quite common, development of PTSD is rare in comparison. Elucidation of the factors responsible for why some people develop PTSD, while others exposed to similar threatening events do not, may inform our understanding of key variables in the etiology of this condition.

Posttraumatic Stress Disorder in the United States Population

The first study to examine the prevalence of PTSD in the general U.S. population was the Epidemiological Catchment Area (ECA) Survey. As measured by the National Institute of Mental Health (NIMH) Diagnostic Interview Schedule (DIS),

the prevalence of lifetime PTSD was observed to be less than 1% of the general population, with higher rates among women than men (Helzer et al. 1987). Scholars criticized this study because of the limited sampling of the population, the manner in which traumatic events were elicited, and the accuracy of the DIS PTSD module (Keane & Penk 1988).

Perhaps the most complete general U.S. population studies are those conducted by Kessler and colleagues. In the original National Comorbidity Survey (NCS; Kessler et al. 1995), a nationally representative sample of 5877 individuals aged 15 to 54 years were interviewed using a substantially revised version of the DIS. An overall lifetime PTSD prevalence rate of 7.8% was found, with rates for women (10.4%) more than twice that for men (5.0%). Trauma exposure estimates indicated that about 60% of men and 51% of women were exposed to one or more traumatic events. In the National Comorbidity Survey Replication (NCS-R; Kessler et al. 2005), a nationally representative sample of 5692 individuals were interviewed using the World Mental Health Survey Initiative Version of the World Health Organization Composite International Diagnostic Interview. Similar to the rates reported in the NCS, an overall lifetime PTSD prevalence rate of 6.8% was found. Gender differences and rates of trauma exposure are not yet published.

Compared with the NCS and NCS-R, similar or slightly higher PTSD rates were obtained in selected, specialized samples. For example, among 21- to 30-year-old members of a Detroit-area health maintenance organization, 40% reported experiencing a traumatic event and 9.5% met PTSD criteria (11.3% of women and 5.6% of men; Breslau et al. 1991). Similarly, among former Miami-Dade public school students aged 18–23, 11.5% met lifetime PTSD criteria (15.5% of women and 7.5% of men; Lloyd & Turner 2003). Further, in two American Indian tribes, lifetime PTSD rates were 14.2% and 16.1%; past year rates were 4.8% and 5.8%, with higher rates consistently found among women than among men (Beals et al. 2005).

The impact of sexual assault and other criminal acts was emphasized in several epidemiological studies focused on documenting PTSD in women. For example, Kilpatrick et al. (1992), in a nationally representative sample of women, learned that 13% of 4008 participants reported a completed rape. Of those who were raped, lifetime and current PTSD rates were 32% and 12%, respectively. Similarly, using a national probability sample, Resnick et al. (1993) estimated that 36% of women had been criminally victimized, with 14.3% experiencing attempted rape or molestation and 12.7% experiencing a completed rape. They estimated lifetime and current PTSD rates to be 12% and 5%, respectively. Among those who were exposed to criminal victimization, rates of lifetime and current PTSD were 26% and 10%, respectively.

Clearly, the prevalence of exposure to traumatic events in the United States is far more common than anticipated in 1980 when the diagnosis of PTSD was incorporated into the diagnostic nomenclature. Even more surprising are findings indicating that the rate of current PTSD in the general population falls only behind major depression, attention-deficit/hyperactivity disorder, specific phobia, and social

phobia, making it the fifth most common psychiatric condition in the United States (Kessler et al. 2005).

Posttraumatic Stress Disorder Among United States Combatants

Soldiers whom we send to fight wars and to keep peace are among those most at risk for trauma exposure and the development of PTSD. Despite the high frequency of military action and war worldwide, few countries have ever comprehensively estimated the psychological toll of war. The major exception to this was the National Vietnam Veterans Readjustment Study (NVVRS; Kulka et al. 1990), which included a representative sample of 1632 U.S. Vietnam Theater Veterans (VTVs), a matched sample of 716 Vietnam Era Veterans (VEVs), and 668 civilian comparison subjects. Sixty-four percent of VTVs were exposed to a traumatic event in their lives, compared with 48% of VEVs and 45% of civilians. More than 15% of male VTVs and 9% of female VTVs met criteria for current PTSD, and 30% of male VTVs and 27% of female VTVs met criteria for lifetime PTSD. Notably, the direction of this gender difference is opposite that of the above-reviewed civilian samples, which is likely attributable to the different roles women had in the military at that time, the different types of stressors to which they were exposed, and their higher educational levels. In all cases, PTSD prevalence rates for VTVs were many times higher than were those found for the VEVs and civilians.

Peacekeeping has its own set of stressors associated with it, including instructions to not fire unless fired upon and orders to patrol areas that are often under tenuous control. Litz et al. (1997) examined a sample of 3461 active-duty peacekeeping military troops who served in Somalia. Shortly after their return to the United States, 8% of these soldiers reported PTSD, a rate that did not differ for men and women. Eighteen months after their return, 6.5% of a subsample of 1040 veterans met criteria for delayed-onset PTSD (Gray et al. 2004).

Several studies examined the impact of service in recent wars in the Persian Gulf. For instance, Wolfe et al. (1993) conducted a longitudinal study of 2344 Gulf War I veterans and found PTSD prevalence rates of 4% for men and 9% for women. Studies using smaller convenience and reservist samples found PTSD rates in the range of 16% to 19% (Perconte et al. 1993, Sutker et al. 1993). Among soldiers deployed during Operation Iraqi Freedom and Operation Enduring Freedom, Hoge et al. (2004) found that 6.2% of the Army soldiers met screening criteria for PTSD after deployment to Afghanistan, and 12.9% met criteria after deployment to Iraq. Among the Marine Corps soldiers deployed to Iraq, 12.2% met screening criteria for PTSD.

Given the notably high prevalence rates of PTSD among combatants, as well as findings indicating that PTSD symptoms among a subgroup of Vietnam veterans do not remit (Schnurr et al. 2003), continued study of combatants is a national priority. Further substantiating the chronic course and nature of combat-related PTSD are findings obtained from the detailed study of the military's testing of mustard gas

on soldiers during World War II. Although few knew of this testing until the 1990s, fully 32% of those exposed continued to exhibit full PTSD symptomatology a half century later (Schnurr et al. 2000).

Posttraumatic Stress Disorder Across the Globe

With the increasing recognition of the health and economic costs associated with psychological morbidity across the world (Murray & Lopez 1996), there is a growing acknowledgment of the need for regional and world estimates of psychiatric disorders, including PTSD.

Economically developed countries report a wide range of PTSD prevalence rates, perhaps owing to differences in culture, language, experiences, and study methodologies. The lowest rates of PTSD were found in Iceland among a cohort of half the birth population in the year 1931 (i.e., lifetime rate of 0.6%, with no men meeting criteria; Lindal & Stefansson 1993). Among a representative sample of 10,641 Australian adults, past-year PTSD prevalence was estimated at 1.5%, with similar rates among women and men. Among the 57% of the sample who experienced a trauma, higher rates were found among women (3.8%) than men (2.0%), potentially because experiencing rape or sexual molestation were the traumas most likely to result in PTSD (Creamer et al. 2001, Rosenman 2002). Among male Australian Gulf War I veterans, 5.4% were found to have developed PTSD after the war, whereas only 1.4% of a comparison sample of veterans who did not deploy to the Gulf developed PTSD during that time (Ikin et al. 2004).

PTSD prevalence rates in less economically developed countries tend to be higher than in more developed countries. For example, among a geographically diverse sample of Mexican adults, lifetime PTSD prevalence was estimated to be 19% (Norris et al. 2003). In addition, among adult Israeli residents, Bleich et al. (2003) found that 9.4% met criteria for current PTSD, with higher rates among women (16.2%) than among men (2.4%). Finally, in a Palestinian sample, Punamaki et al. (2005) found PTSD prevalence rates of 21.5% and 13.2% for men and women, respectively.

Substantially higher rates of PTSD are observed in those countries that are non-Western and developing. Many of these estimates were derived following periods of war and political turmoil. An examination of large, fairly representative samples of men and women age 16 or older living in Algeria, Cambodia, Ethiopia, and Gaza, de Jong et al. (2001) found high rates of PTSD in each sample (37.4%, 28.4%, 15.8%, and 17.8%, respectively). In Algeria and Cambodia, and consistent with findings in the United States, women had higher rates of PTSD than did men (43.8% versus 32.2% and 34.2% versus 20.6%, respectively). In contrast, in Ethiopia and Gaza, women possessed similar or lower rates of PTSD in comparison with men (15.2% versus 16.6% and 13.5% versus 22.6%, respectively).

In a representative sample of Kosovar Albanians age 15 or older assessed approximately one year after the end of the 1998–1999 war in Kosovo, Cardozo et al. (2003) observed a PTSD prevalence rate of 25%, compared with the rate of 17% that was found immediately following the war (Cardozo et al. 2000). Similarly,

immediately following the siege of Sarajevo, Rosner et al. (2003) found that 18.6%
of residents of Sarajevo met criteria for PTSD.

After the bombing raids launched by the United States on Afghanistan and
following a long history of war, conflict, and drought, Cardozo et al. (2004) studied
a representative sample of disabled and nondisabled Afghan people age 15 or older.
Similarly high rates of PTSD were found among the disabled and nondisabled
samples (i.e., 42.2% and 43.1%, respectively). Women in both groups exhibited
higher rates of PTSD than did men, findings that were attributed to the widespread
restrictions placed on women during the Taliban regime. Near this same time,
and using a similar methodology, Scholte et al. (2004) found that 20.4% of persons
age 15 or older in the Nangarhar province of eastern Afghanistan met criteria for
PTSD, with much higher rates found among women (31.9%) than among men
(7.5%).

In a sample of Rwandans living in four diverse communes eight years after the
1994 genocide in that country, Pham et al. (2004) reported a 24.8% prevalence rate
of PTSD, with higher rates again found among women than men. Interestingly,
participants in this study reported particularly high rates of avoidance symptoms.
These findings were discussed in terms of the Rwandan cultural discouragement
of open displays of emotion.

According to De Girolamo & McFarlane (1996), one of the most significant
limitations of the PTSD literature is a lack of research on persons in developing
countries, particularly since they appear to be at elevated risk for experiencing
trauma. These researchers reported that only 6% of PTSD prevalence studies were
conducted in developing countries. Although international studies of trauma con-
tinue to grow, much more work is necessary. Clearly, it is unlikely that research
conducted in the United States and other developed countries can be readily gener-
alized to the developing world. Differences in violence, disasters, cultures, social
structures, and coping behaviors may significantly influence the prevalence and
course of PTSD among people of different countries (De Girolamo & McFarlane
1996). Perhaps the most important conclusion one can draw from these interna-
tional data, however, is that PTSD is represented in similar ways across cultures,
languages, racial/ethnic groups, and geographic areas. The growing numbers of
studies conducted across continents that verify the presence of PTSD as a func-
tion of exposure to traumatic events suggest that PTSD is more than a Western
construction and likely has common biological and psychological underpinnings
irrespective of the many differences inherent in the world's population.

Posttraumatic Stress Disorder Among Refugees

Fazel et al. (2005) summarized the data across multiple studies, including a total of
5499 adult refugees resettled in Western countries. They found substantial variabil-
ity in prevalence rates (range = 3% to 44%). When restricting analyses to more
rigorously designed studies including at least 200 participants, the average PTSD
prevalence rate was 9%. This conclusion is different from that of De Girolamo &
McFarlane (1996), who summarized 12 studies of refugees, and reported that half

of the studies had a PTSD rate equal to or higher than 50%. Several factors likely account for such variability in PTSD rates across studies, including the nature of the sample and the trauma experienced, the length of time since the trauma, and the lack of PTSD measures validated among refugee samples (Hollifield et al. 2002, Keane et al. 1996).

Posttraumatic Stress Disorder Following Disasters

A burgeoning literature suggests that a wide range of natural and man-made disasters can lead to the development of chronic PTSD. Green and colleagues found that 44% of survivors of the collapse of the Buffalo Creek Dam in West Virginia in the late 1970s met criteria for PTSD, and 28% of the sample still met diagnostic criteria 14 years later (Green et al. 1990a,b). Similarly, McFarlane (1989) studied the effects of Australian bush fires on a sample of firefighters. PTSD prevalence rates were estimated at 32%, 27%, and 30%, respectively, at four, eleven, and twenty-nine months post disaster.

In recent years, concern regarding the psychological consequences of terrorist attacks has increased substantially. In a review of studies reporting PTSD prevalence rates following terrorist attacks, Gidron (2002) reported a mean rate of 28%. However, these studies varied greatly with respect to sampling and the timing of assessments.

Following the September 11, 2001 terrorist attacks on the World Trade Center, telephone and Web-based methodologies were employed to examine levels of PTSD symptoms throughout the United States. Rates of PTSD generally were higher based upon regional proximity to the attacks. For example, Schlenger et al. (2002) found the prevalence of probable PTSD to be 11.2% in the New York City metropolitan area, but much lower in Washington, DC (2.7%), other metropolitan areas (3.6%), and the remainder of the country (4.0%). In a sample of adults residing in an area of Manhattan closest to the World Trade Center, 7.5% reported symptoms consistent with a PTSD diagnosis, and 20.0% of a subsample residing closest to the World Trade Center reported such symptoms (Galea et al. 2002).

These are just a few of the many different types of disasters examined to date. Overall, the epidemiological literature strongly suggests that various types of disasters contribute substantially to the development of PTSD.

A CONDITIONING MODEL OF THE ETIOLOGY OF POSTTRAUMATIC STRESS DISORDER

Although we do not know the cause of PTSD, identification of the precipitating event or proximal cause is relatively simple. Keane & Barlow (2002) proposed a triple vulnerability model of PTSD etiology based on theoretical descriptions of anxiety and fear. The three components of vulnerability are pre-existing psychological variables, pre-existing biological variables, and the experience of a traumatic event. Unlike specific phobia, where true alarms, false alarms, or (less often) simple

transmission of information may develop into a simple phobic reaction, PTSD is hypothesized to emerge from one special chain of events. Intense basic emotions, such as true alarms (but also including rage, disgust, or distress, resulting from the overwhelming effects of traumatic events), lead to learned alarms. Learned alarms occur during exposure to situations that symbolize or resemble an aspect of the traumatic event, such as anniversaries of the trauma and thoughts, feelings, and memories (i.e., cues) of the event. As in any phobic reaction, the development of learned alarms can result in persistent avoidance of stimuli associated with the trauma. These are defining features of PTSD.

Wirtz & Harrell (1987) provide support for the process of classical conditioning in PTSD. They observed that victims of physical assault were less distressed six months after the assault if they had experienced exposure to situations or stimuli that were part of (or resembled) the context of the original assault without experiencing another assault. Survivors who had not had the advantage of this exposure, on the other hand, maintained a high level of distress in the six-month interim. Others note the importance of conditioning in the development of PTSD (e.g., Keane & Kaloupek 1982, Keane et al. 1985), whether the trauma is combat related or involves physical assault such as rape (e.g., Holmes & St. Lawrence 1983, Kilpatrick et al. 1985).

The pure experience of alarm or other intense emotions is not sufficient for the development of PTSD. Much as in other disorders, one must develop anxiety or the sense that these events, including one's own emotional reactions to them, are proceeding in an unpredictable, uncontrollable manner. When negative affect emerges, anxious apprehension and preoccupation with the traumatic event begins and collateral PTSD symptoms appear.

The presence and severity of the various PTSD symptoms may be moderated to some extent by coping skills and social support. In PTSD, evidence already exists that these variables play a prominent role in determining whether the disorder develops. These factors are represented explicitly in the model presented in Figure 1; further elaboration of potential risk (or vulnerability) factors for PTSD is presented below.

RISK FACTORS FOR POSTTRAUMATIC STRESS DISORDER

Although many people experience traumatic events, most do not develop PTSD; individuals who experience the same or a similar stressor may or may not develop the disorder. The identification of the factors that might account for these differences can assist in our understanding of the etiology of PTSD and in the development of preventative approaches for people who are at highest risk for developing the disorder. Potential PTSD risk factors can be divided into three major categories: (*a*) pre-existing factors specific to the individual, (*b*) factors related to the traumatic event, including one's immediate response during the trauma, and
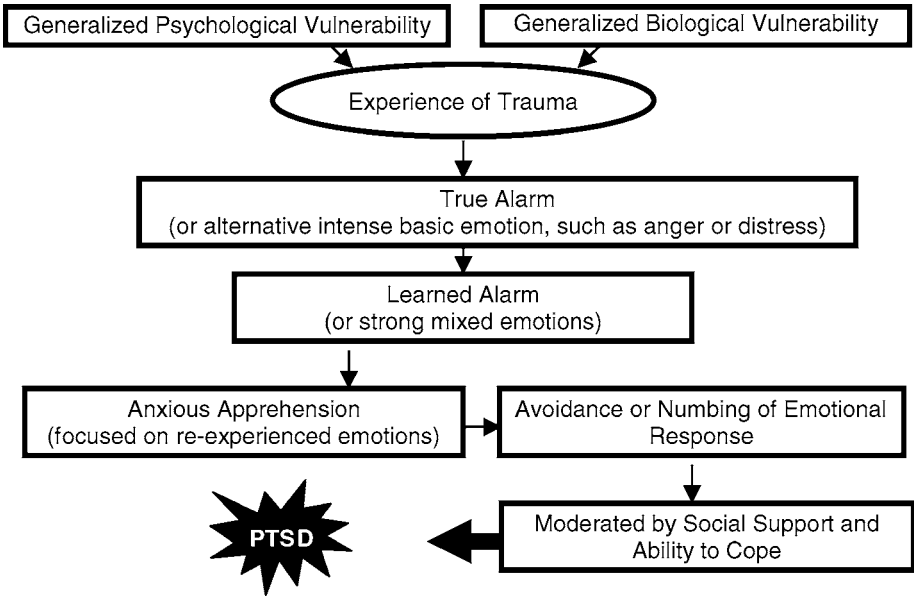
Figure 1   A conditioning model of the etiology of posttraumatic stress disorder (PTSD).

(*c*) events that occur following the trauma. Although the latter category may not be considered "causal" risk factors, such variables may help us better understand the delay in the development of PTSD and the commonly found severe, chronic cases of PTSD (Bromet et al. 1998).

Pre-existing Factors

Discriminating the predisposition for developing PTSD from the predisposition for exposure to traumatic events represents a methodological and interpretive challenge. This issue is profoundly important for accurately identifying those variables that contribute to the development of PTSD. We begin to address this issue by examining pre-existing factors that are related to trauma exposure and the development of PTSD.

FAMILIAL PSYCHOPATHOLOGY   Early fear-conditioning research in animals suggests that there may be a genetic component associated with variability in sensitivity to environmental stress (e.g., Anisman et al. 1979). Although studies of such a mechanism in humans do not exist, several twin and family studies have examined the heritability of PTSD.

The Vietnam Veteran Twin Registry was used to observe heritability factors for exposure to combat (Lyons et al. 1993, True et al. 1993) and PTSD symptoms (True et al. 1993). Despite an apparent genetic link for experiencing certain classes of traumatic events (Koenen et al. 2002, Stein et al. 2002), genetic influences on

trauma exposure do not appear to be shared with those influencing the development of PTSD when the trauma is combat (Roy-Byrne et al. 2004). For noncombat interpersonal violence, there appears to be some association between genetic influences on trauma exposure and genetic influences in the development of PTSD (Stein et al. 2002). Unfortunately, this study used questionnaire data, thus deferring strong conclusions. In perhaps the most elegant twin study of genetic contributions to the development of PTSD, Pitman and colleagues (2002) used a wide variety of laboratory tasks (e.g., physiological reactivity, evoked response potential) and standardized diagnostic tools in an attempt to elucidate the parameters that might underlie PTSD. They found little evidence of an inherited component for PTSD among Vietnam combat veterans.

To help clarify conflicting results, Ozer et al. (2003) recently applied meta-analysis to nine twin and family studies. They concluded that, overall, family history of psychopathology predicted a small but significant amount of variance of PTSD (average weighted effect size of $r = 0.17$). Interestingly, family history was more strongly related to PTSD when the traumatic experience involved noncombat, interpersonal violence than when it involved combat or an accident. Similarly, in their earlier meta-analysis of 11 studies examining family history of psychopathology, Brewin et al. (2000) found an average weighted effect size of $r = 0.13$, with the same effect found in military and civilian samples. These effect sizes are small, and it appears that unique environmental contributors to the development of PTSD are significantly stronger than any genetic influences measured to date (McLeod et al. 2001).

The results of twin and family studies demonstrating genetic factors associated with PTSD suggest that genes influencing this risk may be identified. However, only a few genetic marker studies have been conducted and there are, unfortunately, inconsistent results. Investigators examined allelic associations at the D2 dopamine receptor gene, as it has previously been associated with other psychiatric disorders. Comings and colleagues (Comings et al. 1991, 1996) found associations between the D2 dopamine receptor gene and PTSD, but Gelernter et al. (1999) did not. Thus, despite fairly consistent findings of a small association between familial psychopathology and the development of PTSD, the actual mechanisms accounting for this association (either genetic or environmental) are far from clear.

DEMOGRAPHIC FACTORS

Gender Early epidemiological studies found relationships between demographic variables and exposure to trauma and between demographics and the development of PTSD. For example, we know that PTSD prevalence varies as a function of the type of trauma experienced, and we know that men are more likely to be exposed to a traumatic event during their lives, yet we also know that women are more likely to develop PTSD. Some have hypothesized that this heightened rate of PTSD is a function of the types of events to which women are exposed (e.g., sexual assault). However, even when controlling for gender differences in types of traumas, women appear to be more at risk for developing PTSD than are men (Breslau et al. 1999;

Kessler et al. 1995). Similar gender differences in PTSD prevalence rates have
been found in a variety of samples (e.g., Kosovar Albanians; Cardozo et al. 2003),
yet the average weighted effect size across studies is small ($r = 0.13$; Brewin et al.
2000), and elucidating the mechanisms (i.e., psychological or biological) involved
in these differences surely requires additional scientific study.

Age   Age at the time of a traumatic event is frequently seen as an important
determinant of response, with the very young and the very old carrying additional
risk for disorder upon exposure. Interestingly, among women in the NCS, no
relationship existed between age and PTSD, and only a small positive relationship
between age and exposure to traumatic events was found. In contrast, among men,
there was a strong positive correlation between age and PTSD, but this was due to
increasing exposure to traumatic events over the life span (Kessler 1995). Across
29 studies, Brewin et al. (2000) found that the average weighted effect of age on
PTSD was minimal ($r = 0.06$).

Race   The data regarding race as a risk factor are complicated by great variability
across datasets. For example, in the NVVRS (Kulka et al. 1990), PTSD prevalence
rates were highest among Hispanic veterans, and higher among African American
veterans than the aggregate of Caucasian, Asian, and Native American veterans.
These findings were largely, but not entirely, due to differences in rates of combat
exposure. In the NCS, Kessler et al. (1995) compared racial/ethnic groups and
found that Caucasian and Hispanic participants reported higher rates of trauma
exposure, whereas African American, Asian American, and Native American par-
ticipants reported higher rates of PTSD following such exposures. Using clinical
interview data from the Hawaii Vietnam Veterans Project, Friedman et al. (2004)
reported that veterans of Japanese ancestry had lower odds of a current PTSD
diagnosis compared with Caucasian veterans from the NVVRS dataset even after
adjusting for age and combat exposure. Finally, in a sample of American Indian
Vietnam veterans, Beals et al. (2002) found that the American Indian sample
had higher rates of current and lifetime PTSD than Caucasian participants of the
NVVRS. Yet, when exposure to atrocities and violence was included in a mul-
tivariate model, ethnicity no longer predicted current or lifetime PTSD. Overall,
racial/ethnic status does not appear to provide consistent differences in PTSD
prevalence, and this variability across studies may be because race/ethnicity is
not a strong predictor of PTSD. Importantly, Brewin et al.'s (2000) meta-analysis
found that the effect of racial/ethnic status as a predictor of PTSD was small ($r =
0.05$), with larger effect sizes found for SES ($r = 0.14$) and education ($r = 0.10$),
variables often associated with racial/ethnic status in Western societies.

Marital status   Very few large-scale studies have examined the relationship of
marital status to PTSD prevalence. In the NCS (Kessler et al. 1995), marriage
appeared to confer some level of protection when one was exposed to a traumatic
event even when holding trauma exposure constant in the analyses. In contrast, in

Breslau et al.'s (2004) Detroit trauma survey, marital status was not significantly associated with PTSD after controlling for the type of trauma that the participants rated as worst.

Overall, similar demographic variables have been associated with the development of PTSD across languages and cultures. In studies conducted in Afghanistan (Scholte et al. 2004), Rwanda (Pham et al. 2004), Kenya (Njenga et al. 2004), Turkey (Basoglu et al. 2004), Mexico and Poland (Norris et al. 2002), and Australia (Creamer et al. 2001, Rosenman 2002), comparable risk factors emerge in the analyses. The variability in the predictive relationships is often attributed to demographic factors serving as proxies for variables such as familial and social burdens and access to resources within various countries (Norris et al. 2002). As with other predictors of PTSD, the mechanisms for these associations are not yet known.

PRIOR TRAUMA AND LIFE ADVERSITY   Increasing evidence suggests that prior life trauma and cumulative adversity may increase risk of PTSD following a later trauma. Brewin et al. (2000) found small effect sizes for childhood abuse ($r = 0.14$), other previous trauma ($r = 0.12$), and other adverse childhood factors ($r = 0.19$). Ozer et al. (2003) also conducted a meta-analysis of prior trauma and found a small but significant effect size ($r = 0.17$). This effect did not vary based upon the time elapsed since the trauma or whether the trauma occurred in childhood or as an adult. However, the effect size did vary according to whether the prior trauma resulted from an accident, combat, or noncombat interpersonal violence ($r = 0.12$, $r = 0.18$, and $r = 0.27$, respectively). Interestingly, ethnic groups often differ in terms of distal and proximal life adversity, and variability in life adversity appears to decrease the relationship between ethnicity and PTSD, which suggests that race/ethnicity and other demographic differences may be markers for histories of unequal levels of life adversity (Lloyd & Turner 2003).

Together, these studies suggest that prior trauma and life adversity may sensitize people to later traumas. With a large degree of variability across study samples, it is possible that some individuals (e.g., nurses, firefighters) develop adaptive coping skills that may protect them from adverse responses to future traumas. Further, Bowman & Yehuda (2004) suggested that if normal stress hormones (e.g., cortisol) are activated over prolonged periods of time, brain physiology and anatomy may be altered, leading to a depletion of hormones that results in an inadequate physiological response to later trauma exposure. More research on this as a possible biological mechanism is clearly needed.

PSYCHOPATHOLOGY PRIOR TO THE TRAUMA   Several studies identified the prior existence of a psychiatric condition as a risk factor for the development of PTSD. In both Brewin et al.'s (2000) and Ozer et al.'s (2003) meta-analyses, psychiatric history was found to confer a small degree of risk for the development of PTSD ($r = 0.11$ and 0.17, respectively). However, when Ozer et al. examined a subset of studies that specifically investigated prior depression, the degree of risk increased significantly ($r = 0.32$).

The presence of psychopathology such as an addictive disorder or conduct disorder may also lead to exposure to traumatic events themselves (Breslau et al. 1991, Kessler et al. 1995). This complex relationship is fundamental to our understanding of the effects of traumatic events and PTSD. Careful assessment of the precipitating variables that contribute to a particular psychological condition can provide meaningful information about which condition to treat first when intervening with patients with multiple comorbid psychological disorders (e.g., Najavits 2000).

In a fascinating prospective study of risk for developing PTSD, Bryant & Guthrie (2005) measured maladaptive cognitions of student firefighters during their training and prior to deployment. Pretrauma catastrophic thinking strongly predicted (24% of the variance) the level of PTSD symptomatology 20 months after training was completed, indicating that a tendency to catastrophize about aversive events is a risk factor for the eventual development of PTSD.

The Traumatic Event Itself

Characteristics of the event itself, not surprisingly, predict the development and severity of PTSD. Most theoretical models suggest the presence of a dose-response model of PTSD in which symptom severity is a function of traumatic event severity. Yet, operationally defining severity for various traumatic events is a complex task. Peritraumatic variables measured to date include a range of factors such as physiology, affect, and cognitions that occur during the trauma, as well as particular aspects of the type of traumatic event.

Trauma Severity

Numerous studies defined the severity of a traumatic event in a variety of creative ways and found a significant association with PTSD severity. For example, across 49 studies, Brewin et al. (2000) found an average weighted effect size of $r = 0.23$ for the association between trauma severity and PTSD severity. In addition, Norris et al. (2002) suggest that severity is a strong predictor of PTSD cross culturally.

Some studies examine proxies for traumatic event severity and relate them to severity of PTSD. For example, among survivors of the Oklahoma City bombing, suffering physical injuries was strongly related to PTSD symptoms six months later (Tucker et al. 2000). In addition, in an Australian national sample, Rosenman (2002) found that experiencing combat and rape or molestation were events that were especially likely to increase one's odds of developing PTSD. Finally, in a sample of Mexican adults, Norris et al. (2003) found that exposure to violence in childhood was related to the chronicity of PTSD.

PERCEIVED LIFE THREAT AND PERITRAUMATIC EMOTIONAL RESPONSE   Across 12 studies, Ozer et al. (2003) found a small to medium weighted effect size ($r = 0.26$) for the strength of the relationship between perceived life threat and PTSD. Interestingly, the strength of the relationship was higher in studies with more time

elapsed between the traumatic event and the assessment of PTSD. Moreover, in a recent study, perceived life threat was associated with maintenance of PTSD symptoms over time (Schnurr et al. 2004).

Ozer et al. (2003) separately examined the relationship between peritraumatic emotional response (e.g., fear, helplessness, horror, guilt, shame) and PTSD and the found a similar effect size across five studies ($r = 0.26$). Notably, Tucker et al. (2000) suggested that feeling nervous or afraid, expecting to die, and being upset by how others reacted during the Oklahoma City bombing explained 67% of the variance in PTSD symptoms six months later. Peritraumatic emotional distress also appears to predict the chronicity of PTSD symptoms (Norris et al. 2003). Interestingly, in their study of Somalia peacekeepers, Gray et al. (2004) found that negative perceptions of the mission were associated with immediate and chronic posttraumatic symptomatology.

Individual differences in peritraumatic emotional response may be due to differences in the meaning assigned to stressful events. Dunmore et al. (2001) found that cognitions measured shortly after the trauma, such as negative beliefs about oneself and the world, were risk factors for PTSD severity six and nine months later, even after controlling for the severity of the event. As reviewed by Bowman & Yehuda (2004), several retrospective and cross-sectional studies found relationships between cognitive beliefs and PTSD severity. Such cognitions included negative appraisals of trauma symptoms, low self-efficacy, and external locus of control.

PERITRAUMATIC DISSOCIATION   Peritraumatic dissociation includes symptoms such as an altered sense of time, "blanking out," and feeling disconnected from one's body (Marmar et al. 1994). Across 16 studies, Ozer et al. (2003) found a medium weighted effect size ($r = 0.35$) for the strength of the relationship between peritraumatic dissociation and PTSD. Although the strength of this relationship did not differ according to the type of trauma experienced, it did vary as a function of the time elapsed between the trauma and symptom measurement, the type of sample, and the method of symptom assessment. Peritraumatic dissociation was also found to play a role in the maintenance of PTSD (Schnurr et al. 2004).

To better understand this relationship, Gershuny et al. (2003) examined the role that cognitions of panic (i.e., fears of death and/or losing control) might play in the association between peritraumatic dissociation and PTSD. They found that retrospectively reported fears of death and losing control during the trauma mediated the relationship between peritraumatic dissociation and PTSD. They suggest that peritraumatic dissociation might be part of the panic process, and the cognitive elements of panic may override the dissociative elements in their relative importance for PTSD development. These researchers emphasize that these cognitions may elicit dissociation and that their interaction might be responsible for the observed relationships.

Halligan et al. (2003) studied survivors of a recent physical or sexual assault. They found that peritraumatic dissociation may lead to the disorganized trauma

memories that are characteristic of PTSD. They also found that degree of disorganization in trauma memories and negative appraisals of that disorganization, as well as negative appraisals of intrusive memories, predicted PTSD symptoms over the course of six months. This was true even after controlling for actual memory characteristics and depressive symptoms. Overall, cognitive variables such as cognitive processing during the trauma, trauma memory disorganization, persistent dissociation, and negative interpretations of trauma memories each predicted PTSD symptoms beyond objective and subjective measures of stressor severity.

Clearly, cognitive factors and their relationship to reports of peritraumatic dissociation are important areas for future inquiry. Yet, this work is centered on the accurate measurement of peritraumatic dissociation. For example, Marshall & Schell (2002) found that individuals' recall of peritraumatic dissociation within days of the trauma differed markedly from their recall at three- and twelve-month follow-ups, and baseline dissociation did not predict subsequent PTSD symptom severity after controlling for initial symptom severity. Thus, it is possible that the cognitive factors inherent in Ehlers & Clark's (2000) model and found in the Bryant & Guthrie (2005) prospective study may ultimately prove most important to the prediction of PTSD.

Posttrauma Factors

Very few studies include the assessment of posttrauma factors and examine their contribution to the development and maintenance of PTSD. Among such factors, social support has received the most attention. Across 11 studies, Ozer et al. (2003) found that perceived social support following the traumatic event was associated with PTSD symptoms, with an average effect size falling in the small to medium range ($r = -0.28$). This effect was strongest among studies in which more time elapsed between the trauma and the assessment. Similarly, Brewin et al. (2000) found an average weighted effect size in the medium range ($r = -0.40$) across 11 studies for the relationship between social support and PTSD symptoms. In both meta-analyses, this relationship was stronger among military/combat samples. Furthermore, among Vietnam veterans, Schnurr et al. (2004) found that emotional sustenance and instrumental assistance, both at homecoming and at interview, in addition to structural social support at interview, decreased the odds of having ever had PTSD; all variables except instrumental assistance at homecoming and at interview decreased the odds of chronic PTSD. Recently, King et al. (2006) examined the directionality of the relationship between PTSD and social support among a sample of Gulf War I veterans examined on multiple occasions. Using structural equation modeling and a cross-lagged panel design, they found that PTSD symptoms more strongly predicted subsequent social support than social support predicted subsequent PTSD symptoms. These findings support the idea that interpersonal problems associated with PTSD negatively influence one's support resources. Clearly, further work is

needed to replicate these findings across samples and to examine the nature of this relationship.

Modeling the Prediction of Posttraumatic Stress Disorder

To begin to disentangle associations found in risk factor analyses, multivariate analytic methods such as structural equation modeling (SEM) are used to simultaneously examine interrelationships among several predictors of PTSD. In a series of studies, Daniel and Lynda King and their colleagues (King et al. 1995) applied SEM to a wide variety of theoretically driven models using the NVVRS dataset to understand the relationship of various factors in the prediction of PTSD following the Vietnam War. In the first of their series of studies, war-zone stressor variables of atrocities/abusive violence, perceived threat, and malevolent war-zone environment had direct effects on PTSD, with malevolent environment exerting the largest effect. Traditional combat exposure had an indirect effect, influencing the development of PTSD primarily through the perceived threat that individuals reported (King et al. 1995).

Next, King et al. (1996) examined demographic variables, prewar factors (i.e., family environment, childhood antisocial behavior, maturity at entry to Vietnam, and prior trauma exposure), and war-zone stressor variables as predictors of PTSD. In separate models for men and women, they found that war-zone stressors were important contributors to PTSD, but additional variance was attributable to the prewar factors, particularly for men. Prior history of trauma and age at entry to Vietnam were important factors for men; for women only prior trauma history contributed to the ultimate development of PTSD.

King et al. (1998) then examined hardiness, structural social support, functional social support, and recent stressful life events as possible factors predicting PTSD. When examined together with the war-zone stressor variables, all four variables had direct effects on PTSD development for men. For women, a similar model was revealed, with the slight exception that structural support did not contribute to the prediction of PTSD.

In the final report of their series of studies, King et al. (1999) aggregated the pretrauma risk factors, the war-zone stressors, and the posttrauma resilience-recovery variables to more comprehensively understand how these variables interrelate in the development of PTSD. Remarkably, these three categories of variables predicted 72% of the variability in PTSD among women and 70% among men. Prewar trauma exposure, exposure to abusive violence and life threat during the war, as well as postwar life stressors and functional social support were the strongest predictors of PTSD for women. Among the men, the key variables were the same as for women, plus younger age at entry to Vietnam, the malevolent war-zone environment, and structural social support. Although future studies should examine constitutional, physiological, and hereditary factors, the level of variance accounted for in these studies without the inclusion of biological variables is quite impressive.

TREATMENT OF POSTTRAUMATIC STRESS DISORDER

Contemporary PTSD interventions were largely influenced by prior work in the area of psychoanalysis, particularly that of Janet (1889) and Freud (1936). As with contemporary anxiety management interventions and exposure therapy, psychoanalytic treatment involved attempts to reduce the high levels of anxiety and reactivity to the trauma, and the reconstruction of trauma memories with the accompanying emotional reactions in order to promote a sense of mastery (Fenichel 1945). The inclusion of hypnosis as a viable treatment for traumatized people added a systematic and technical approach to achieving the psychoanalytic objective of reconstructing traumatic memories. Similarly, in the aftermath of World War II, the use of sodium amytal interviews to recreate and recall details of traumatic war events became increasingly widespread. Again, the objective of these interviews was to identify critical elements of traumatic events that were not reported by the patients so that efforts to address the psychological sequelae of these events could begin.

Largely driven by the psychological and social problems of returning American Vietnam veterans, mental health practitioners established programs to treat traumatic disorders for these large numbers of war veterans (e.g., Keane & Kaloupek 1982). Treatment models, initially focused upon conditioning models, have grown to increasingly accommodate behavioral and cognitive factors (Brewin et al. 1996, Ehlers & Clark 2000, Foa et al. 1989, Lang 1977, Litz & Keane 1989), and these models are among the most widely recommended and evaluated today. Derived from these initial models of PTSD, exposure therapy, cognitive therapy, and anxiety management techniques are three of the most accepted and empirically tested treatments for PTSD. A more recent approach to treating PTSD is eye-movement desensitization and reprocessing (EMDR; Shapiro 1989). This is essentially an atheoretical technique that was accidentally discovered to alter disturbing thoughts, feelings, and images (Shapiro 1995). It has been applied to a broad range of psychological problems, but the bulk of the available research on its efficacy is with PTSD.

This section on treatments for PTSD briefly describes and examines the supporting evidence for psychological therapies, including exposure therapies, anxiety management training (AMT), EMDR, and combination therapies. Pharmacological interventions are also reviewed in this section. Other treatments for PTSD are in developing phases and/or do not have strong empirical evidence supporting their use and as a result we do not include them in this review.

Empirically Examined Psychological Interventions for Posttraumatic Stress Disorder

EXPOSURE THERAPIES   In exposure therapy for PTSD, the patient is guided through a vivid remembering of the trauma until extinction occurs. This therapy is applied using in vivo and imaginal forms of exposure. In vivo exposure generally involves

returning to the site of the traumatic event to reduce avoidance and promote mastery over the associated trauma cues. For example, if a person developed PTSD following a car accident, therapy might include returning to the scene of the accident with the patient, perhaps with the patient driving (Blanchard & Hickling 1997). Imaginal exposure is often used when in vivo exposure is not possible. Cues are presented in imagery in an effort to describe details of an event or set of events from the perspective of the stimulus propositions, response propositions, and meaning propositions associated with the event (Lang 1977). Similar to in vivo exposure, the goal of imaginal exposure is to reduce avoidance and promote mastery.

ANXIETY MANAGEMENT TRAINING   Typically, AMT involves teaching patients an assortment of behavioral and cognitive strategies to enhance their capacity to manage the emotions associated with PTSD. Such skills might include relaxation training, breathing retraining, trauma education, guided self-dialogue, cognitive restructuring, and communication skills training. Some programs for PTSD have emphasized the incorporation of anger management training as a part of the skills taught to patients (Chemtob et al. 1997, Keane et al. 1989), given the salience of anger difficulties among patients with PTSD (Novaco & Chemtob 2002).

EYE-MOVEMENT DESENSITIZATION AND REPROCESSING   EMDR entails imaginal exposure to traumatic images while the patient moves his/her eyes horizontally or vertically back and forth, tracking the therapist's index finger. Negative cognitions are also evoked and the patient is instructed to focus on more positive thoughts. This procedure is repeated until the patient no longer reports distress. Although some discussion of Pavlovian neurophysiology is provided across publications (Shapiro 1989, 1995), the mechanism of action for EMDR is not based on any contemporary theories of human behavior, learning, or cognitive science. For this reason, it has been criticized in the scientific literature (Herbert & Mueser 1992; Herbert et al. 2000; Keane 1998; Lohr et al. 1995, 1998; McNally 1999).

COMBINATION TREATMENTS   Combination treatments that make use of an array of cognitive behavioral strategies may be helpful in addressing the multiple problems that people with PTSD might exhibit. Keane et al. (1994) described a PTSD treatment package that included exposure therapy, AMT, and cognitive restructuring as central features of their approach. This package employs a phase-oriented approach to treating severe and chronic PTSD that includes six phases: (*a*) behavioral stabilization, (*b*) trauma education, (*c*) anxiety management skills training, (*d*) trauma focus work, (*e*) relapse prevention skill, and (*f*) aftercare procedures.

Resick & Schnicke (1992) developed a multidimensional behavioral treatment package originally developed for women with rape-related PTSD, but more recently applied to a range of other trauma groups. This package, entitled cognitive processing therapy (CPT), combines elements of exposure therapy, cognitive therapy, and AMT. The exposure component of CPT involves writing and reading

about the traumatic event. The cognitive therapy component of CPT involves addressing key cognitive distortions resulting from the traumatic event. Specifically, difficulties in safety, trust, power and control, self-esteem, and intimacy are targeted.

Clinical Studies of Psychological Interventions for Posttraumatic Stress Disorder

A number of recent reviews and meta-analyses provide strong support for the efficacy of psychological interventions for PTSD (Bradley et al. 2005, Davidson & Parker 2001, Hembree & Foa 2000, Sherman 1998, Van Etten & Taylor 1998). Much of the early clinical research on PTSD was conducted among military veterans. Keane et al. (1989) compared exposure therapy to AMT and to a wait-list condition for the treatment of combat-related PTSD in Vietnam veterans. Relative to wait-list participants, patients receiving imagery-based exposure therapy showed reductions on therapist ratings of PTSD symptoms, as well as self-reported depression, anxiety, and fear, at posttreatment. Most of these changes were maintained at the six-month follow-up.

Several additional studies reported the salutary effects of exposure therapy for combat veterans with PTSD. Boudewyns & Hyer (1990) and Cooper & Clum (1989) demonstrated that the addition of exposure therapy to available treatments for PTSD improved outcome for patients. Glynn et al. (1999) assessed the effects of adding a family-based skills training intervention to imaginal exposure therapy. Exposure therapy resulted in declines in symptoms of anxiety, arousal, and reliving the trauma relative to a wait-list comparison group, and these treatment gains were maintained at a six-month follow-up. Notably, the addition of family behavioral treatment did not enhance the efficacy of the exposure therapy intervention.

Some recent work has examined the efficacy of exposure therapy and AMT-based interventions for refugees. Paunovic & Ost (2001) found that both of these interventions were highly effective in reducing PTSD symptoms, anxiety, and depression, and these gains were maintained at follow-up. Neuner et al. (2004) demonstrated the efficacy of an exposure-based intervention for PTSD among a sample of traumatized Sudanese refugees. Specifically, narrative therapy, which is a form of exposure therapy that involves the extinction of emotional responding to traumatic reminders and the construction of one's autobiographical narrative, was compared with psychoeducation and supportive therapy in a randomized trial. Large differences were found between the study groups. Only 29% of those that received narrative exposure therapy continued to meet criteria for PTSD at a one-year follow-up, compared with 79% and 80% rates for the supportive therapy and psychoeducation groups, respectively.

Considerable research has examined the efficacy of PTSD interventions for victims of interpersonal violence, sexual assault, and abuse. Foa et al. (1991) compared exposure therapy, an anxiety management condition called stress inoculation, supportive counseling, and a wait-list condition in the treatment of rape-induced

PTSD. Measures included clinical ratings of PTSD symptoms and standardized psychometric inventories, all administered at pretreatment, posttreatment, and a three-month follow-up. Findings indicated that both active treatments were superior to the control conditions in reducing PTSD at posttreatment, and the stress inoculation treatment evidenced the greatest effects. However, over time exposure therapy demonstrated more reductions in PTSD than did stress inoculation.

More recently, Foa et al. (1999) compared exposure therapy with AMT as well as a combination of the two treatments among a sample of assault victims with PTSD. All three of these treatments effectively reduced symptoms of PTSD and resulted in functional improvement relative to a wait-list comparison group. Some evidence suggested the superiority of exposure therapy alone. A subsequent study by Foa & Rauch (2004) indicated that exposure therapy resulted in substantial decreases in PTSD-related cognitions and the addition of cognitive restructuring did not enhance this effect.

Resick et al. (2002) compared CPT and exposure therapy in the treatment of rape-related PTSD. Treatment effects were examined at posttreatment as well as at three- and nine-month follow-up periods. In general, the two treatments were highly and equally effective, and significantly more effective than a wait-list control condition. The active therapies differed only on the reduction of guilt where CPT was superior on two of four measures. Importantly, follow-up analyses further revealed that both active therapies were effective with women who presented with childhood sexual abuse histories and attendant complex symptom pictures (Resick et al. 2003).

Kubany and colleagues (2004) examined the efficacy of a combination therapy that incorporated exposure therapy techniques and various components of AMT and cognitive therapy among a sample of battered women. Relative to wait-list controls, the intervention group experienced significant reductions in PTSD, depression, and guilt, and concomitant increases in self-esteem. Further, 87% of women in the intervention group no longer met criteria for PTSD at posttreatment and therapeutic improvements were maintained at both three- and six-month follow-up assessments.

A large body of research documents the efficacy of PTSD interventions among people reporting heterogeneous trauma histories. In an early study, Brom et al. (1989) examined the efficacy of exposure therapy, hypnotherapy, and psychodynamic treatment among a sample of patients who had experienced a range of traumatic events. Comparable levels of change were noted in each of the active treatment groups and all three groups demonstrated greater change than those in a wait-list condition.

Marks et al. (1998) examined exposure therapy and cognitive restructuring, alone and in combination, among outpatients with PTSD secondary to a wide range of traumatic events. All three active treatment conditions showed significant improvement and greater improvement than that observed in a relaxation therapy comparison group. The three treatments did not differ from one another on the key outcome variables.

Tarrier et al. (1999) compared exposure therapy and cognitive therapy in the treatment of outpatients with PTSD stemming from many different traumatic events. Both treatment groups manifested similar levels of improvement that was maintained at the six-month follow-up period. In a recent five-year follow-up study, Tarrier & Sommerfield (2004) found that treatment effects held up well, though superior long-term outcomes were found for cognitive therapy. The high number of participants lost at follow-up, however, tempered conclusions.

Consistent with earlier work, Bryant et al. (2003) found among a sample of civilian trauma survivors that those receiving exposure therapy and cognitive restructuring plus exposure therapy manifested greater reductions in PTSD symptomatology and anxiety than those receiving supportive therapy. In contrast to some earlier studies, these researchers found that the combination of exposure therapy and cognitive restructuring led to a greater reduction in PTSD symptoms and maladaptive cognitive styles than did exposure therapy alone, suggesting an added benefit of this combined intervention. In a more recent study, a cognitive intervention for PTSD that incorporated elements of AMT and exposure therapy successfully ameliorated symptoms of PTSD (Ehlers et al. 2005).

Bisson et al. (2004) examined the efficacy of a four-session cognitive behavioral intervention (including elements of exposure and cognitive therapy) for PTSD among a sample of patients receiving medical care for physical injury. At a 13-month follow-up, individuals who received this treatment demonstrated significant reductions in PTSD symptoms relative to a standard care condition in which participants received no formal psychosocial intervention.

Some evidence indicates the effectiveness of combination interventions for PTSD among motor vehicle accident survivors. In a randomized controlled clinical trial, Fecteau & Nicki (1999) learned that an intervention consisting of trauma education, relaxation training, exposure therapy, cognitive restructuring, and guided behavioral practice was successful in reducing PTSD at posttest, with further reductions evidenced at a six-month follow-up. More recently, Blanchard et al. (2003) examined an intervention that similarly made use of psychoeducation and both exposure therapy and AMT features, and that also incorporated anger management and couples-based components. This intervention was superior to supportive therapy and a wait-list condition in reducing PTSD symptomatology as well as comorbid major depression and generalized anxiety disorder. These treatment effects were maintained at the three-month follow-up.

In the development of EMDR, Shapiro (1989) conducted a series of single subject cases and open clinical trials that suggested this approach may promote recovery from PTSD. More recently, randomized clinical studies of individuals reporting different forms of trauma demonstrated that participants receiving EMDR responded better than those assigned to wait-list control groups (Rothbaum 1997, Wilson et al. 1995). In a clinic setting, Marcus et al. (1997) found that EMDR, tested in a health maintenance organization, was more effective than standard psychological care in reducing PTSD symptomatology among individuals reporting diverse traumatic events. EMDR was also associated with lower global distress,

anxiety, and depression. A subsequent study showed that these differences were maintained at three- and six-month follow-up assessments (Marcus et al. 2004).

Carlson et al. (1998) compared EMDR to biofeedback-assisted relaxation training and a routine care condition among a sample of combat veterans with PTSD. In this randomized clinical trial, the EMDR group showed greater clinical improvement than that of both comparison groups on self-report, psychometric, and clinician-rated measures of PTSD, anxiety, and depression. Many of these effects were maintained at a nine-month follow-up.

Devilly et al. (1998) compared EMDR, an equivalent intervention without the eye movements, and a control condition in a sample of PTSD-positive Australian Vietnam veterans. Results indicated that both active treatment groups evidenced comparable decreases in PTSD symptoms; there was no apparent benefit to the inclusion of the eye-movement component. Further, by six-month follow-up, no differences in PTSD symptoms were found between the active treatment and control groups. This study contributes to the growing skepticism in the field regarding the value of the eye movements in achieving the outcomes observed using EMDR (see Herbert et al. 2000).

Other studies reported less-than-impressive efficacy for EMDR. Projects by Boudewyns et al. (1993), Jensen (1994), and Vaughan et al. (1994) found only modest effects for EMDR. Some studies observed a worsening of PTSD symptoms at five-year follow-up among those receiving EMDR when compared with matched controls (Macklin et al. 2000). These studies all have significant methodological limitations, but they are comparable in quality and design to many of the projects that provided the initial empirical support for EMDR.

Unfortunately, EMDR has rarely been compared to other active treatments for PTSD in rigorous, controlled clinical trials, and the little research in this area suffers from methodological limitations. A recent study by Taylor et al. (2003) represents an important exception to this. In this investigation, EMDR, exposure therapy, and relaxation training were compared among a sample of persons reporting various forms of trauma and who were recruited from physician referrals and media advertisements. Results indicated that all three interventions reduced PTSD, related depressive symptoms, dissociative symptoms, and trauma-related anger and guilt at posttreatment. Yet, exposure therapy was more effective in reducing avoidance and re-experiencing symptoms and tended to work more rapidly at reducing avoidance. Exposure therapy also was associated with a higher proportion of participants who no longer met PTSD criteria after treatment.

In summary, exposure therapy, cognitive therapy, and interventions incorporating AMT components are treatments that enjoy considerable empirical support in well-controlled clinical trials (efficacy studies) across diverse populations of trauma survivors. Future studies examining the effectiveness of these approaches in clinic settings (effectiveness studies) are warranted. Some evidence also supports the efficacy of EMDR, although findings vary across studies and investigations evaluating this procedure suffer from some methodological limitations. If there is a fundamental weakness of EMDR, it stems from the absence of a

theoretical model, a lack of specification of the mechanisms of change, and a lack of integration with existing psychological models of psychopathology and psychotherapy.

Pharmacological Treatments for Posttraumatic Stress Disorder

PTSD is a complex disorder that involves several neurobiological systems (Bremner et al. 1999, Charney et al. 1993, van der Kolk 1997). Efforts to index a single system are likely to prove to be inadequate in treating a disorder with this level of biological complexity. Yet, recent advances in the understanding of the biology of PTSD highlight the importance of pharmacological interventions (Albucher & Liberzon 2002, Friedman 2000), and accordingly the number of clinical trials of pharmacologic treatments for PTSD has increased rapidly in the past decade.

Antidepressant medications are the most frequently studied pharmacological agents for PTSD and are the most frequently prescribed in clinics. Among the antidepressants, the selective serotonin reuptake inhibitors (SSRIs) are the most typically prescribed pharmacological intervention. Two such medications, sertraline and paroxetine, were approved by the Food and Drug Administration for the treatment of this disorder. Several controlled trials examining the efficacy of SSRIs for the treatment of PTSD indicate that this class of medications is modestly effective in the treatment of PTSD and its associated problems (Brady et al. 2000, Connor et al. 1999, Davidson et al. 2001, Martenyi et al. 2002, Tucker et al. 2001, van der Kolk et al. 1994). Treatment effects are typically manifested across the different PTSD symptom clusters. Further, studies consistently show that these medications are at least as effective as other antidepressants, but are associated with fewer side effects.

Tricyclic antidepressants (TCAs) have been studied less than SSRIs in recent years, likely because of their undesirable side effects and potential toxicity. Some evidence from controlled trials indicates the effectiveness of TCAs, specifically imipramine (Kosten et al. 1991) and amitriptyline (Davidson et al. 1990). Some uncontrolled trials also provide support for the use of TCAs for PTSD (Burstein 1987, Kauffman et al. 1987). However, perhaps due to their side effects, particularly high rates of dropout were reported in studies of TCAs (Kosten et al. 1991).

Some controlled trials indicate the effectiveness of monoamine oxidase inhibitors (MAOIs) for the treatment of PTSD, specifically phenelzine (Kosten et al. 1991) and brofaromine (Katz et al. 1994). Other controlled trials did not support the efficacy of MAOIs (Baker et al. 1995, Shestatzky et al. 1988) and rates of dropout from investigations of these agents were comparable to those of TCAs. A review by Southwick et al. (1994) concluded that MAOIs were more effective than TCAs in the treatment of PTSD. However, MAOIs have received little recent attention because of concerns about their side effects and the requirement that patients taking this medication be placed on a tyramine-free diet and abstain from alcohol and various prescribed and illicit drugs.

Other antidepressants such as nefazodone and trazodone are frequently used to treat symptoms of PTSD, but have not been examined in controlled clinical trials. Trazodone is frequently used as an adjunctive treatment for symptoms of insomnia that often are present among individuals with PTSD (Friedman et al. 2000) and evidence indicates that nefazodone is effective for PTSD-related sleep disturbance (Mellman et al. 1999). One recent controlled trial indicated that mirtazapine was effective in reducing PTSD and general anxiety (Davidson et al. 2003).

Consistent findings from biological studies indicate that individuals with PTSD experience excessive adrenergic reactivity (Southwick et al. 1999), suggesting the potential utility of antiadrenergic agents. These medications have received increased attention recently and one recent controlled clinical trial indicates that prazosin reduced nightmares, improved sleep, and was associated with reductions in overall PTSD symptoms among a sample of PTSD-positive veterans (Raskind et al. 2003). A number of other open trials and field trials suggest the effectiveness of these medications for treating symptoms of PTSD (see Albucher & Liberzon 2002). Some preliminary reports also suggest the possible efficacy of these agents in preventing PTSD when administered in the immediate aftermath of trauma (Pitman et al. 2002). Therefore, antiadrenergic medications may represent an important area of future study for the prevention and the treatment of PTSD.

The use of anticonvulsants for PTSD stems from "kindling" models of PTSD. According to the kindling model, repeated stimulation of limbic structures increases the likelihood of them becoming "sensitized" (Post et al. 1997). One controlled trial (Hertzberg et al. 1999) and several other open trials and case studies suggest the possible effectiveness of anticonvulsants in the treatment of PTSD (see Friedman et al. 2000), though these medications have many potential side effects that make clinical management difficult (Friedman 2003).

Typical antipsychotics have not been examined systematically in the treatment of PTSD and are generally not recommended (Friedman 2003, Friedman et al. 2000). Atypical antipsychotics, which are associated with less-severe side effects, are more promising. Some recent controlled trials demonstrated that augmentation treatment with atypicals led to reductions in PTSD symptoms (Bartzokis et al. 2005, Hamner et al. 2003, Monnelly et al. 2003, Stein et al. 2002), and several open trials and case studies suggest the effectiveness of atypicals (see Ahearn et al. 2003). These medications may be particularly effective when psychotic symptoms, extreme hypervigilance or paranoia, dissociation, and/or intense anger are evident. More research on the use of the atypicals with PTSD is warranted and is ongoing.

In summary, a range of medications is used for treating people with PTSD. The strongest evidence to date is for antidepressant medications, particularly SSRIs. Yet, the effect size for SSRIs even in the largest clinical trials is modest. When compared with the psychological treatments for PTSD, treatment effects in the pharmacotherapy studies are significantly smaller, and serious problems with attrition occur in these medication trials (Van Etten & Taylor 1998). Clearly, there is a need for additional research to further understand the neurobiology of PTSD

so that newer medications can be developed to target the array of neurobiological systems that are dysregulated in PTSD.

CONCLUSIONS

The psychological sequelae of traumatic events are well recognized and PTSD, the most common psychological disorder that follows a traumatic event, is surprisingly prevalent across the world. With the exception of samples of U.S. combatants, higher rates of PTSD are typically found among women than men, both in the U.S. population and worldwide. PTSD prevalence rates also tend to be highest among non-Western and less economically developed countries. These high rates are perhaps due to differences in social structures, including fewer resources following large-scale traumatic events, as well as differences in coping behaviors and culturally driven modes of support. However, study methodology may also play a role in the observation of these differences in prevalence. For example, many studies are conducted in the immediate aftermath of traumatic events, and validated measures of PTSD among the various cultures and samples are not often readily available (Keane et al. 1996).

In recent years, we have begun to make significant progress in the understanding of the etiology of PTSD. Clearly, individual factors play a significant role in determining who may develop PTSD following exposure to a traumatic event. Although pre-existing factors, such as genetic predisposition and demographics, appear to be related to risk for exposure to trauma, they are only modestly related to risk for the development of PTSD; importantly, demographic factors may be associated with PTSD because they serve as proxies for variables such as familial/social burden, access to resources, and life adversity. Indeed, aggregate measures of life adversity and pretrauma psychopathology appear to confer a significant degree of risk for PTSD. However, the greatest risk factors tend to be characteristics of the trauma, peritraumatic response and related cognitions, and posttrauma social support. On the whole, the degree of variability in the development of PTSD explained by psychological factors alone is dramatic. It will be important to continue specifying the mechanisms responsible for these associations [e.g., possibly using Ehlers & Clark's (2000) model] and to further explore physiological or biological factors in ways that can meaningfully inform prevention and intervention efforts.

As data continue to accrue on interventions for PTSD, there is a distinct need for studies to examine combinations of treatments, employ measures that assess social and occupational functioning, and address the impact of treatments on comorbid psychological conditions. Clearly, the available efficacy studies demonstrate the value of extending the use of exposure therapies, cognitive therapies, and AMT to PTSD patients. Evidence indicates that exposure therapy, cognitive therapy, and AMT in their many formats should be given priority by clinicians encountering patients with PTSD. Moreover, the evidence on EMDR suggests that it is likely operating much like other CBT treatments for PTSD that focus on challenging

dysfunctional cognitions in the context of mild levels of exposure therapy (Keane 1998).

With the completion of several well-controlled, empirically rigorous clinical trials of pharmacological interventions for PTSD and with the recent approval of sertraline and paroxetine for the treatment of PTSD by the Food and Drug Administration, the psychopharmacology of PTSD holds much promise for continuing development. As a disorder that affects as much as 8% of the general U.S. population, it is likely that pharmaceutical companies will continue to devote research and development efforts to finding additional and more-effective treatments for PTSD. Clinical trials that further examine the interactive effects of psychological interventions and psychopharmacological agents are clearly warranted in the next generation of treatment outcome studies.

It is also important that preventive interventions for PTSD be further developed and evaluated (Davidson 2004). Some preliminary evidence indicates that psychological interventions (Bryant et al. 1998, 2003, 2005; Ehlers et al. 2003; Foa et al. 1995), pharmacological interventions (Pitman et al. 2002), and combinations of the two (Zatzick et al. 2004) may be effective in preventing the development or progression of PTSD symptomatology. Given our growing knowledge of trauma, PTSD, and its sequelae, such interventions may become more feasible and ultimately may have a substantial payoff with respect to reductions in human suffering.

Worldwide, the problems associated with war, criminal assault, and disasters (natural and technological) are not abating. As a result, sound public policy is needed to guide society's response to survivors of these experiences. PTSD in its most chronic form is a debilitating condition that affects individuals, their families, their communities, and their nations. Those who are the targets of violence may ultimately become perpetrators, thus contributing to the cycle of violence initially documented by Widom (1989). If this is so, then interventions to address the occurrence of violence (primary prevention) or to mitigate its effects once it occurs (secondary prevention) are needed. Reliance upon sound empirical work to devise and implement these prevention efforts may ultimately be the best solution to the problems associated with PTSD.

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